

Fatty Liver in Hispanics with HIV

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EDITOR: Liver disease is a leading cause of non-AIDS-related mortality in persons infected with human immunodeficiency virus (HIV). In this regard, nonalcoholic fatty liver disease (NAFLD) is a growing problem.¹ NAFLD is present in HIV-infected persons with lower body mass indices (BMIs) than in HIV-negative persons.² Among different ethnic populations, Hispanics have a higher incidence and a more aggressive course of NAFLD, even in those with lower BMIs.³ Most studies performed to date have been cross-sectional in patients on antiretroviral therapy (ART) or in Caucasian cohorts with a Hispanic subpopulation.⁴ We performed a small prospective cohort study in Argentina, recruiting 59 consecutive HIV-infected persons before initiating ART. The cohort had an average (\pm SD) age of 36 ± 10 years, 67% (40/59) were men, and 12% (7/59) were coinfecting with either hepatitis B virus or HCV. The median CD4

count was 43 (IQR 20–123) cells/ μ L with 80% (47/59) having CD4 < 200 cells/ μ L. Using the ultrasound score (USS) to quantify fatty liver, all performed by the same radiologist, we found a very high proportion, 66% (37/56), of fatty liver with the majority of individuals ($n = 31$, 55%) having grade 1, mild steatosis, by the USS. Five participants (9%) had grade 2, moderate steatosis, and one participant (2%) had severe steatosis. Moreover, 10 of 59 (17%) persons had an aspartate aminotransferase to platelet ratio index (APRI) score suggestive of fibrosis (>0.8). However, the APRI score did not correlate with findings of fatty liver on ultrasound. The mean pre-ART baseline level for AST was 38 IU/liter and that for alanine aminotransferase was 36 IU/liter. Follow-up evaluations occurred in 39 participants at 6 weeks and 31 participants at 12 weeks. Immune recovery was appropriate with a median CD4 of 108 (IQR, 43–178) cells/ μ L at 6 weeks and

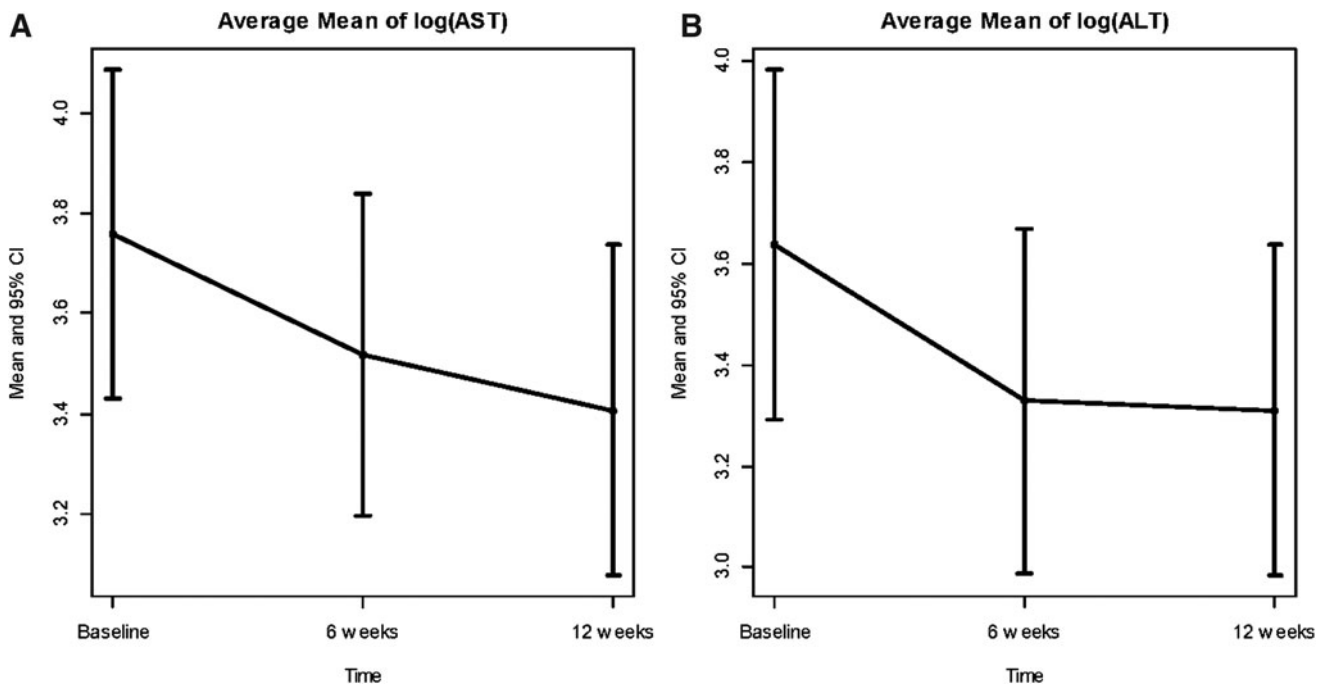


FIG. 1. Decrease in levels of AST (A) and ALT (B) after antiretroviral therapy. AST, aspartate aminotransferase; ALT, alanine aminotransferase.

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153 (IQR, 52–246) cells/ μ L at 12 weeks. Liver transaminases decreased at 6 and 12 weeks post-ART (Fig. 1). Multivariate analysis was performed to examine change of AST and ALT over time using linear mixed model adjusting for baseline CD4, HCV or HBV infection, and alcohol consumption. Due to skewed distribution, AST and ALT were natural log-transformed in the analysis, and back transformed to geometric mean. The geometric mean level of AST decreased from 43 (95% CI 31–60) IU/liter to 34 (95% CI 24–46) IU/liter ($p=.005$) at 6 weeks and to 30 (95% CI 22–42) IU/liter ($p=.0004$) at 12 weeks of ART. The mean level of ALT decreased from 38 (95% CI 27–54) IU/liter to 28 (95% CI 20–39) IU/liter ($p=.009$) at 6 weeks and to 27 (95% CI 20–38) IU/liter ($p=.003$) at 12 weeks of ART. There was no evidence of clinically apparent liver toxicity at 12 weeks of ART in our cohort. We found no association between alcohol consumption, HBV surface antigen (HBsAg) status, and AST, ALT levels. Persons with HCV had an overall log of AST and ALT of 0.75 and 0.52 higher than those who were HCV-negative ($p=.0008$ and $p=.01$, respectively). We found no difference in AST or ALT based on whether or not persons received an ART regimen that included nucleoside reverse transcriptase inhibitor. Interestingly, using a linear mix model, we compared the CD4 count recovery after initiation of ART and found that the drop in AST and ALT correlated with CD4 increase and the correlation was significant for ALT ($p=.005$). Due to factors beyond our control, we were not able to assess and compare HIV viral load. Our study needs further confirmation and steatosis evaluation beyond noninvasive methods. Moreover, our study did not have a control group (HIV-negative or HIV without transition

to ART). However, the results suggest that the rate of steatosis in purely Hispanic HIV-infected patients with low CD4 counts might be higher than expected.

Author Disclosure Statement

No competing financial interests exist.

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